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Expectancy for Outbreaks of Poliomyelitis in Camps and Schools: When a case of poliomyelitis occurs in a camp or a school, (1) is it likely that additional cases will occur, and if so, how many and when, (2) should the school or camp continue to operate, and (3) if it closes, will the dispersed group constitute a significant health hazard to other communities?

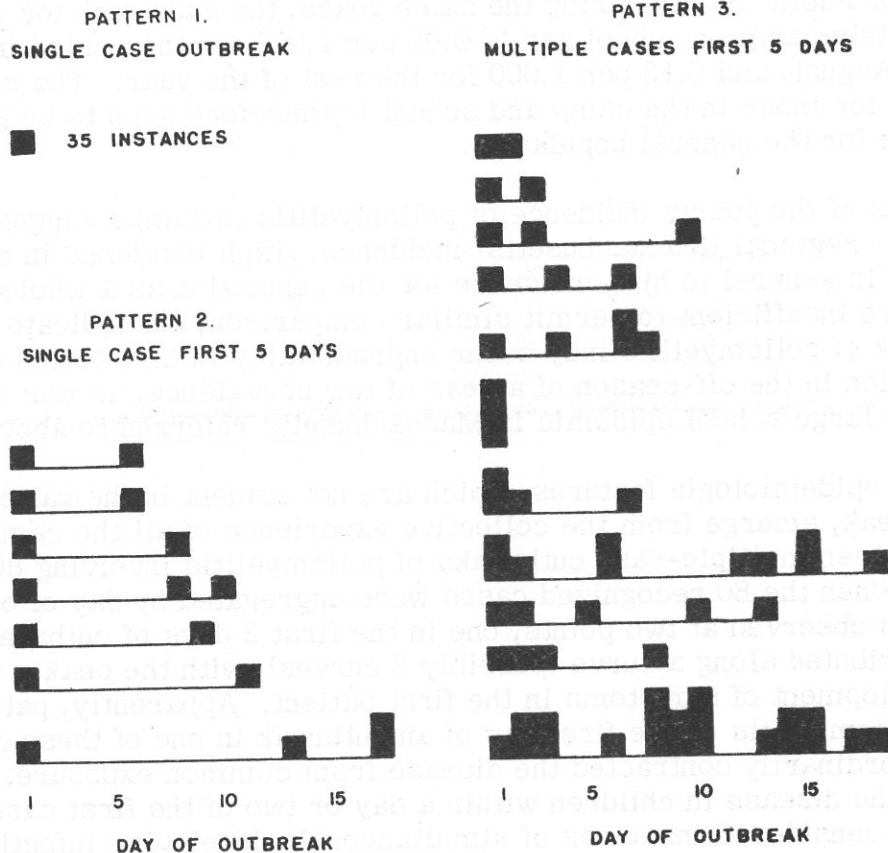
A study was made of the actual behavior of poliomyelitis as it occurs during the summer season in camps and during the rest of the year in boarding schools to determine whether the epidemiologic data may point the way to logical administrative control. An appraisal of the risk of acquiring clinically recognizable disease and knowledge about the probable extent of spread is of practical concern to the physician in charge of a camp or school.

Data were obtained from a questionnaire answered by heads of 140 boys' and girls' camps in New England, and by physicians responsible for the health of 40 representative boarding schools in the northeastern United States. The completed form showed how many times poliomyelitis had appeared and the dates of onset for all cases of the disease occurring in the camp or school since 1935. Then, by letter, telephone call, or visit, this information was cross-checked and further extended through the records of the 6 state health departments in New England. The reliability of clinical diagnoses is good, because most outbreaks were investigated by health officers and clinicians, and most patients had the advantage of hospital study.

During the 14 years from 1935 through 1948, poliomyelitis occurred in 30 (21 percent) of these 140 camps, and in 12 (30 percent) of these 40 boarding schools. Two camps and 2 schools reported that the disease had appeared twice. Infection was usually limited to a single recognized case. No poliomyelitis was reported among campers in 110 (79 percent) of the camps with annual enrollments averaging 94 boys or girls. Twenty-one, with an average attendance of 115, reported single-case outbreaks in those same 14 years, and 11, with an average enrollment of 99 campers, had multiple-case outbreaks. The occurrence of only 11 multiple-case outbreaks among the 140 camps during nearly 2,000 summers of operation is indicative of the rarity of clinically manifest group infection. Of the 40 boarding schools, 28, with an average population of 309 students, reported no case of poliomyelitis among students during the 14-year period. Ten schools, with an average enrollment of 339, had experienced 11 single-case outbreaks. During a total of 560 academic years of operation, multiple-case outbreaks of poliomyelitis were seen on only 3 occasions.

In order to augment data on patterns of spread, 4 additional outbreaks are included in the figure on the next page, one in a camp during 1923, another in a school during 1931, and 2 camp outbreaks which occurred during the summer of 1949. The complete data for 1949 have not yet been collected and analyzed, but so far it appears that the incidence of poliomyelitis in camps during

Patterns of Single and Multiple-Case Outbreaks of Poliomyelitis in 140 Camps
and 40 Schools, 1923-1949.



the summer of 1949 surpassed that of any year for which data have been obtained. One multiple-case outbreak is known to have occurred at one of the 40 schools during October, 1949.

During the period studied, the largest camp epidemic occurred in 1943, a year of high poliomyelitis incidence, and involved 10 (8.2 percent) of 122 boys. The most extensive school outbreak occurred in Massachusetts in 1936, a year of low incidence among the rest of the state school population. Five cases developed within 3 days, initiating a chain of infections involving 21 (11 percent) of 190 boys; that year only 11 cases of poliomyelitis were reported among some 350,000 other Massachusetts children aged from 10 to 14. During the 9 years, from 1940 through 1948, camp cases averaged about 7 per 2 months summer period, and school cases a little more than 1 per academic year. Based upon the camp population, 13,480, and the school population, 12,315 children of susceptible age, the average case rates for the 9-year period are 0.56 per 1,000 campers per summer and 0.13 per 1,000 students per academic year. Using population data for Massachusetts from the U. S. Bureau of the Census for 1940, and the average number of cases of poliomyelitis

reported for persons of from 10 to 14 years of age to the Massachusetts Department of Public Health during the same years, the case rate for children of approximately camp or school age is 0.67 per 1,000 for the period from July through August, and 0.13 per 1,000 for the rest of the year. The risk of poliomyelitis for those in the camp and school is therefore seen to be almost precisely that for the general population.

Analysis of the yearly incidence of poliomyelitis in camps suggests some relationship to regional (Massachusetts) incidence. High incidence in camps corresponded in general to high incidence for the population as a whole. Data for schools are insufficient to permit similar comparison, but indicate that a large outbreak of poliomyelitis may occur unpredictably in an isolated segment of the population in the off-season of a year of low prevalence, as was demonstrated by the large school epidemic in Massachusetts referred to above.

Certain epidemiologic features, which are not evident in the pattern of any one outbreak, emerge from the collective experience of all the camps and schools. Eighteen multiple-case outbreaks of poliomyelitis involving 80 children were noted. When the 80 recognized cases were aggregated by day of outbreak, clustering was observed at two points, one in the first 3 days of outbreak, and the other distributed along a curve (possibly 2 curves), with the peak a week after the development of symptoms in the first patient. Apparently, patients developing poliomyelitis on the first day of an outbreak in one of these closed communities ordinarily contracted the disease from common exposure. Development of the disease in children within a day or two of the first case may be reasonably considered instances of simultaneously developing infection. It may be inferred that patients who develop poliomyelitis within the first 5 days of an outbreak have contracted it from a common source, whereas those who develop the disease later represent secondary attacks among contacts of the initial group of patients. Infection in these secondary cases presumably takes place one or more days before primary or other common source cases are recognized and the patients isolated.

As seen in the figure on the preceding page, 3 patterns of infection are recognized, single-case outbreaks which did not progress, single-case outbreaks which were followed by from 1 to 3 additional clinical cases after an interval of 5 days, and outbreaks of multiple initial cases in the first 5 days followed by as many as 16 cases during the subsequent period of from 2 to 3 weeks. The distinctive fact emerging from the data as a whole is the failure of poliomyelitis to propagate in a clinically manifest form in the large majority of instances in which a single recognized patient presumably exposed the group. In the 7 single-case outbreaks which were followed by cases of recognizable infection after an interval of 5 days, the incidence of secondary attacks did not exceed more than 1 percent of the group exposed. On the other hand, almost half of the outbreaks initiated by multiple-cases occurring within 5 days of each

other resulted in secondary cases constituting from 2 to 8 percent of the exposed group.

The data permit a provisional estimate of the likelihood of spread when poliomyelitis is introduced into closed community groups. If but one case develops during the first 5 days of an outbreak, the likelihood of significant spread is small; in over 80 percent of such outbreaks no further cases may be anticipated. One or two more additional clinical cases may be expected in perhaps 20 percent of such outbreaks, but epidemic spread is unusual. The expectation is different when the outbreak of poliomyelitis is initiated by multiple cases, which indicate that common source infection of several members of the group has occurred. Thus, in these data, when 3 of the 5 outbreaks were ushered in by 2 common source cases, at least one other individual developed infection; in 5 of the 6 outbreaks which began with 3 or more cases, spread of the infection occurred and reached epidemic proportions on 2 occasions. The extent of such spread was generally related to community dosage of the virus as measured by the number of common source cases developing in the first 3 or 4 days of the outbreak. It may be concluded that the prevalence of poliomyelitis in camps and boarding schools corresponds in general to that of boys and girls of the general population. Under existing circumstances a multiple-case outbreak is to be expected in the ordinary camp or school about once every century. (Am. J. Pub. Health, May '50, T. H. Ingalls and A. D. Rubenstein)

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Study on Gastric Biopsy Specimens Obtained Through the Flexible Operating Gastroscope: Since the first successful biopsy of the stomach made by means of the flexible operating gastroscope in 4 March 1948, 63 specimens have been taken for biopsies with no complications and no accidents. The flexible operating gastroscope originally designed was of rather large caliber for general use, being 17.5 mm. at its largest diameter. In a few instances it was impossible to pass it, because of its large size. The new operating gastroscope is 15 mm. at its largest diameter; it is more flexible than the first model and easy to pass. Biopsy has proved useful in the diagnosis of lymphoma, carcinoma, and gastritis. Gastrosopic biopsy is useful in differentiating between lymphoma of the stomach, a surgical problem, and gastritis, a medical problem. A gastrosopic biopsy that demonstrates lymphoma offers positive proof of the disease; a gastrosopic biopsy that does not demonstrate lymphoma may exclude lymphoma with reasonable certainty, because this growth is generally regarded as a diffuse process.

The diagnosis of carcinoma of the stomach may be established beyond any question in a difficult case by a positive gastrosopic biopsy. A biopsy that does not reveal the presence of carcinoma does not exclude a diagnosis of malignant lesion, however, because carcinoma may be a localized process and the specimen may have been taken from an adjacent noninvolved area.

The 63 specimens so far obtained with the flexible gastroscope have been reported by the pathologist as showing normal stomach (18), chronic gastritis (24), acute and chronic gastritis (10), inadequate specimen (6), carcinoma (3), and lymphoma (2).

Although the gastroscopic diagnosis of gastritis based on the gross appearance of the stomach as seen through the flexible gastroscope has become widely accepted, a microscopic report is of assistance in putting the diagnosis of gastritis on a positive histologic basis. The study of biopsy specimens is also of value in correlating clinical, radiologic, gastroscopic and pathologic observations in gastritis. Such studies have so far been based almost entirely on specimens resected for peptic ulcer or carcinoma.

Gastroscopic biopsy may (1) substantiate the gastroscopist's gross impression of gastritis, (2) demonstrate the presence of gastritis in a stomach which appears endoscopically normal, or (3) fail to reveal pathologic evidence of gastritis in a stomach which appears grossly abnormal to the gastroscopist. In addition to furnishing pathologic proof of the presence of gastritis, gastroscopic biopsy is certain to furnish data by means of which one may (1) study the different types of gastritis, (2) correlate the clinical, radiologic, gastroscopic, and pathologic observations in gastritis, and (3) study the relationship of gastritis, ulcer, and tumor.

Benedict and Mallory consider that superficial gastritis as described by the gastroscopist (reddening, edema, and adherent secretion), probably corresponds to acute gastritis as described by the pathologist. Although 63 cases is not a large series, it is already evident that many cases of superficial gastritis are actually chronic in the opinion of the pathologist. Presumably, however, acute gastritis will be found unaccompanied by chronic gastritis in some cases.

Hypertrophic gastritis is reported by the gastroscopist when he sees verrucous elevations in the mucosa, sometimes with segmentation of the rugae and a dull appearance of the mucosa, with diminished highlights. Such an appearance usually corresponds to chronic gastritis as described by the pathologist. Acute changes may be superimposed. It is evident from biopsy specimens already obtained that chronic gastritis may be found pathologically when both radiologic and gastroscopic examinations fail to reveal it. It will be interesting to determine by gastroscopic biopsy whether or not most persons addicted to the consumption of alcoholic liquors actually do have some degree of gastritis. Extensive intestinal metaplasia, as seen histologically, is one of the indications of gastric atrophy. Although there was some intestinal metaplasia in 4 cases in this series, in all of which chronic gastritis was also shown, no biopsy has been reported as demonstrating gastric atrophy. The mucosa is so thin in complete gastric atrophy that one hesitates to attempt a biopsy for fear of perforation. Postoperative gastritis is sometimes regarded as a separate entity, but there is no gastroscopic or pathologic evidence to support this theory.

The correlation of clinical, radiologic, gastroscopic, and pathologic observations in gastritis will require careful analysis in hundreds of cases before any conclusions are justifiable. Evaluation is difficult. Gastritis may be present in patients without symptoms, whereas symptoms resembling those in gastritis may be present in patients not having gastritis. Peptic ulcer similarly may exist without symptoms, and ulcer-like symptoms may be present in ulcer-free patients. If symptoms consistent with gastritis are present and the gross gastroscopic picture is definitely that of gastritis, but the biopsy specimen shows a normal gastric mucosa, one should rely on the observation of a trained gastroscopist, because the biopsy specimen may have been taken from a relatively normal area in the midst of other areas which would have revealed gastritis.

The relationship between gastritis, ulcer, and cancer is not clear. Most observers agree that some gastritis accompanies every ulcer and every tumor, but differ in their opinions concerning the causative role of gastritis in ulcer or tumor. The author has never been able to demonstrate that gastric ulcer developed in an area of gastritis. Neither has it ever been proved that carcinoma develops from gastritis, although there is some evidence that adenomatous polyps and carcinoma are more common in gastric atrophy. It is possible that frequent gastroscopic biopsy may give useful material for study of the precursors of ulcer and tumor.

In 6 cases (10 percent) in this series, the biopsy specimen has been considered inadequate. The decision as to adequacy is left entirely to the pathologist. (Arch. Path., May '50, E. B. Benedict)

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Diagnosis and Treatment in Celiac Disease: From the authors' experience with 603 cases of celiac disease a useful method of diagnosis of this confusing condition has emerged together with an effective cure by diet which has been found successful in cases of all types and degrees of severity.

Celiac disease is altered intestinal function characterized by stools, which are more frequent than usual and altered in physical characteristics. Varying degrees of disturbance of nutrition may be present, depending upon the duration and severity of the condition, and upon the diet. The disease may be protracted or intermittent, and it occurs independently of other known causative factors such as specific infections, parasitic infestations, or abnormal anatomy.

Once this altered intestinal function exists, it will continue as long as the ingestion of carbohydrates, other than those found in fruits and to a lesser extent in vegetables, continues. A diet free of all carbohydrates except those in fruits and vegetables results in the disappearance of symptoms; symptoms

will recur when forbidden carbohydrates are again ingested. If, however, the diet containing only carbohydrates in fruits and vegetables is continued for a sufficient length of time, there will be no recurrence of stool abnormalities or nutritional disturbance when forbidden carbohydrates are added.

The chief symptom of celiac disease is diarrhea, which may exist from birth or may begin at any time thereafter, usually within the first few years of life. The stool may be watery, but is usually only soft and mushy, more voluminous than in health, with frequency of from one to 10 daily. It is sometimes oily, often mucoid, and usually foul. The color varies from pale cream to greenish yellow. Each attack of diarrhea may last a few days, weeks, or months, but there are intervals when stools return to normal. The recurrence of diarrhea with such intervals of almost normal stools is very characteristic and a most valuable diagnostic feature. In rare cases there is constipation, making diagnosis difficult. The second most common symptom is irritability with whining and crying, coupled with weakness and asthenia, and usually anorexia. These symptoms are the first to disappear with correct treatment, usually within a week or two. Next in frequency among symptoms is the failure to gain weight and to grow. In more severe cases there is actual loss of weight. The extreme emaciation so prominently mentioned in classical descriptions of celiac disease occurs only in the most severe cases. The large abdomen of the classical picture is similarly found only in advanced cases; it varies in size from day to day and, representing a real anatomic change, outlasts all other symptoms. Common symptoms of celiac disease are abdominal pain and vomiting. Colic is more frequently encountered than vomiting, but both are commonly found up to the time when correct treatment is begun, after which they soon disappear. Other symptoms are subsidiary, and depend upon the malnutrition and the extent of deprivation of specific dietary factors; among these are anemia, edema, hydrolability, enlarged heart, photophobia, rachitic manifestations of varying degrees, hemorrhagic states, and pareses.

Celiac disease is often unrecognized in its early stages, because its primary symptoms are so general and unspecific. The disease usually begins insidiously, sometimes as a sequel to acute gastro-intestinal disturbance or an infection. The bowel disturbance may begin so gradually that it is not taken seriously for some time.

The incidence of celiac disease is difficult to estimate; however, it is much more common than was formerly thought. It spares no group; the wealthy, the poor, the well-fed, the starving are equally subject to it. The average physician sees so few patients with celiac disease that many may pass him unrecognized. Celiac disease may occur at any age, although it is encountered most commonly among children under 6 years of age. Evidence exists which suggests that in adults the condition is identical with nontropical sprue. Although many investigators have stated flatly that celiac disease never occurs in

breast-fed infants, the authors' records and the literature show many such cases. Celiac disease may exist from birth, and it is not at all uncommon under one year of age.

Some evidence supports the view that there is a familial tendency toward the disease, but more than one case is seldom found in the same family at the same time.

The cause of celiac disease is unknown. All pathologic abnormalities found in celiac disease are encountered also in other conditions. Some investigators believe that a disturbed pancreatic function is significant, but analysis of duodenal secretions does not contribute essential information.

The only definite method of diagnosis consists of the response to the correct diet, better termed specific carbohydrate diet, described herein. If immediate beneficial results follow the institution of the diet, the case may well be considered one of celiac disease. If forbidden carbohydrates are then introduced into the diet of such a patient who is doing well, and an attack of loose stools results, this is practically pathognomonic, especially if reinstitution of the specific carbohydrate diet controls the diarrhea. This method of diagnosis was used in the 603 cases in this report, and the 370 patients seen over a long period received a treatment based primarily upon the specific carbohydrate diet.

One basic principle of the diet must be established firmly and reiterated persistently: no food may be ingested by the patient that contains an appreciable amount of carbohydrate other than that found in fruits and to a lesser extent in vegetables, and in protein milk. Although this principle may be easily understood, it is difficult in practice always to recognize the existence of the carbohydrates in various foods. The basis of the specific carbohydrate diet is ripe banana and protein milk. When the child will not drink protein milk, calcium

*Protein milk prepared according to Finkelstein and Meyer:¹⁹ One quart milk, warmed to temperature of 98° F. To this is added 1 tablespoon of essence of pepsin. Allow to drain through cheesecloth for one-half hour to separate the whey from the curd. The curd mixed with 1 pint of water, is then rubbed through a fine wire strainer several times, and to it 1 pint of buttermilk is added. The whey which contains most of the sugar is discarded.

Protein milk as prepared by Mueller and Kran:²⁰ Mix 1 quart of buttermilk (commercial) and 1 quart of water and heat to a temperature of 135° F. Remove from the stove and let stand for one-half hour. The curd by this time is well separated from the whey, 36 oz. of which should be dipped off. The remaining curd and whey are mashed through a fine sieve, and 4 oz. of 20 per cent cream or 4 oz. from top of bottle of milk, and enough water added to bring the mixture to 32 oz.

Powdered protein milk: 12 tablespoons of the powder to 32 ounces of water.

Calcium caseinate milk: Use 4 to 6 tablespoons of calcium caseinate (Mead Johnson's Casec) to 1 pint of water and 1 pint of milk. Mix the Casec with a little cold water (enough to form a smooth paste), pour in the remainder of the cold water. Then pour in the milk and bring the whole mixture to a boil while stirring constantly, then boil actively for one minute. Remove from fire. Let cool. If necessary to sweeten, use one or two tablets of saccharin (1 gr.).

caseinate milk may be used. Protein milk should be prepared in one of the 3 ways described in the small print on the left. Banana is the most satisfactory and the only safe fruit to be used at the outset of treatment. It is a 20-percent carbohydrate and thus replaces better than any other fruit the excluded carbohydrates; it has a very low fiber content, is easily obtainable, palatable, and well liked by most children. It may be served raw or baked. Only fully ripe bananas should be given, with no trace of green at the tips, the skin well speckled with brown, and the edible portion soft enough to mash easily. If ripe bananas are not available or practical to use, banana powder which is exactly equivalent to fully ripe bananas

may be substituted, or dried banana flakes may be given, although these are not quite as satisfactory as fresh ripe bananas. Because other fruits have qualities which tend to make them laxative, they must be employed judiciously when diarrhea is still active. Most canned fruits are forbidden because of added sugar. If cooked fruits are desired, they may be prepared with saccharin by the family.

The specific carbohydrate diet, in addition to protein milk and fruits, may contain proteins in any form and fats in moderate quantities. Thus meat, fish, and fowl of any kind may be used, and it is not necessary or even advisable to have all the fat removed. All cheese is satisfactory, unless it has been processed by the addition of ingredients to alter the composition. Gelatin is given for dessert in this diet, but not prepared gelatin desserts which contain sugar. Desserts made from pure gelatin, fruit juice, and saccharin for sweetening are well tolerated; honey, dates, and raisins may be used as confections, because the sugars in them are levulose and glucose, but some dates are packed in sugar syrup to make them adhere in one mass, and these should not be used.

When brisk diarrhea is controlled, egg is added to the diet. When the stools are formed and occur no more than 2 or 3 times daily, vegetables are given, but they must be added to the diet cautiously, one at a time with a sufficient period between each new introduction to determine the effect. In some cases diarrhea recurs when vegetables are ingested, in which case their use must be postponed. In general, lettuce, tomato, string beans, squash, and carrots are well tolerated. Canned vegetables are not used because many have sugar added. Potato may not be used.

Fats in association with meats in the normal amounts, in the form of butter, and that existing in protein milk are well borne. Sour cream is usually tolerated. When a full and well-rounded diet has been established, there need be no restrictions of fat beyond that usually exercised in the diet of healthy children.

With these foods, the specific carbohydrate diet is complete. Because it is full and well-balanced, it is continued for at least one year, supplemented by certain vitamins. Aqueous soluble vitamins A and D should be administered. One of the preparations of B complex, including folic acid, seems to be desirable. Some form of iron should be given for the anemia.

In prescribing this diet, it is almost more important to stress what is not fed than what is fed. Any cereal grain is strictly and absolutely forbidden, including corn, wheat, rye, or rice in any form, whether as bread, cake, toast, zwieback, crackers, cookies, or breakfast cereals. Potato is prohibited. Sugar is forbidden as sweetening or in the form of candy, pastries, breads, etc., as

well as dextrans such as are found in corn syrups and lollypops. Milk, other than protein or calcium caseinate milk, is not allowed.

The strictness of this diet cannot be overemphasized, nor should the difficulty of adhering to it be minimized. Faithful observance requires intelligence and vigilance on the part of the mother or the person taking care of the child with celiac disease.

At the beginning of treatment, the patient is put on a so-called basic diet:

Breakfast: pot cheese, bananas, protein milk. Lunch: meat, pot cheese, bananas, protein milk. Supper: the same as lunch; gelatin may be added to any meal. Any of these foods may be used in any quantity or given between meals. After one week, orange juice, other cheeses, and egg may be added, one at a time and with a sufficient interval to test the acceptability of each. After 2 weeks all fresh fruits may be tried in the same way. When stools are controlled, vegetables (except potato) may be added; sometimes they are well tolerated but often their introduction must be postponed. Tolerance is eventually attained, at which time the diet is complete. If, at the beginning of treatment, there is clinical evidence of gastro-intestinal hyperactivity such as colic or vomiting, as is frequently the case, this may be controlled by suitable doses of atropine. Most patients begin to improve immediately. The earliest sign of improvement is a change in the child's disposition; he becomes happy, smiling, contented. Diarrhea is often controlled in the first week, but in some cases such control may take a few months. Appetite improves if there has been anorexia, and the patient begins to grow and gain weight. During the first 6 months or more, any infection, especially in the upper respiratory tract, may be accompanied by a recurrence of the diarrhea. Also in this period, the ingestion of a forbidden carbohydrate will bring about loose stools within hours or days, but the attack will quickly subside if no more of the forbidden carbohydrates are ingested. A break in the diet after about 6 months will not usually be reflected immediately in diarrhea, but the ingestion of forbidden carbohydrates must be continued for some time, even weeks, for diarrhea to recur. Otherwise, there are no relapses or so-called crises or catastrophes such as those described in much of the literature on the subject, requiring therapy to combat acidosis and dehydration.

Duration of treatment is of utmost importance. The strict diet must be continued for at least one year. If there has been no recurrence of symptoms, forbidden carbohydrates may be added: one slice of bread 3 times daily, or a bowl of cereal once and bread twice; or cereal at breakfast, toast for lunch, and spaghetti for supper. If these additions to the diet cause no diarrhea, then potato is given. After 3 months with no disturbance, plain milk is added, and if no diarrhea occurs in the next 3 months with these additional foods, the patient

may be considered cured, and all restrictions on the diet are lifted. The entire cure usually requires no more than 18 months, but when the diet has not been rigorously followed, it may take a much longer time. When cure is obtained there should be no relapse. A striking example of the persistence of the disease in a case in which the proper diet was not followed is a patient who was treated up to the age of 6 years. The proper regimen was not followed strictly because her mother owned a candy store and it was impossible to keep the patient from forbidden carbohydrates. At the age of 22 years she returned with all the symptoms she had had in childhood, and which had existed through the years. Placed on a strict diet, which she followed faithfully, all symptoms soon disappeared. Now, 18 months later, she is apparently cured, although not yet on a full diet.

Among milder cases of celiac disease there is a degree of tolerance for carbohydrates which allows for careless treatment with fair results, but cure requires 2 or 3 times as long as would be the case if a strict diet were followed. In some such cases the symptoms are little relieved but physical progress is maintained so as to obscure the fact that a cure has not been obtained. Fortunately, time eventually seems to help these patients to get well, although many of them go through life with a tendency to loose stools.

This diet differs, in varying degrees, from all those previously advocated in the literature on the subject. The basic difference is that the authors' diet excludes all carbohydrates except those in fruits, in some vegetables, and in protein milk. The proper type of carbohydrate to be fed is of the utmost importance, for experience shows that even the smallest quantity of the forbidden carbohydrates will precipitate diarrhea.

Standard works on celiac disease restrict the use of fat, ascribing the cause of the disease to both fat and carbohydrate intolerance or to fat intolerance alone. The evidence behind such beliefs is the frequent occurrence of steatorrhea in the disease. In the authors' experience fat does not incite diarrhea. When the whole dietary is low, fat must be somewhat restricted as it would be in the feeding of normal children; when proteins and carbohydrates derived from fruits are increased in the diet, fat may be taken in usual quantities. The absence of fat from the diet is probably a contributory cause of rickets in many cases reported. The only satisfactory kind of protein milk is that prepared according to one of the 3 methods given earlier in this article, or calcium caseinate milk, in all of which the sugar content is low. All others should be avoided.

Although the dietary regimen described above was the basis of treatment in the patients in this study, other modes of therapy have been considered and some of them tried. It has been found that injections of vitamin B complex and liver extract will in many cases end diarrhea for a varying period of time; but

diarrhea usually recurs when such injections are stopped. This treatment is painful and disliked by patients. The use of antibiotics is usually followed by cessation of diarrhea, but it will recur after a period even while the antibiotic is still being given. Pancreatic extract has not often been used by the authors because dietary treatment gives the desired results. However, there are rare cases in which the addition of pancreatic extract seems to help.

Under the dietary treatment described, prognosis is excellent. Practically all patients recover and there should be no deaths.

A most important prognostic conclusion from the cases reported here is that in most cases cure can be effected within 18 months, and the acceptance of a slight diarrhea for 4 or 5 years until the child "outgrows it" is fallacious and harmful to the patient. With proper therapy pulmonary involvement is unusual and rarely fatal, and there need be no stunting or permanent mark of the disease. No avitaminosis was seen in any of the authors' cases; rickets did not occur, chiefly they believe, because fats were not excluded from the diet.

The 603 children in whom the diagnosis of celiac disease was made have been seen by the authors over a period of some 25 years. Of this total, 233 were not seen frequently enough or for a sufficiently long period to evaluate either the therapy used or the results. Of the 370 patients adequately followed, 270 (73 percent) were cured, 89 (24 percent) were recent and on the road to cure, 8 (2.2 percent) were not cured, and 3 (0.8 percent) had died. Cured patients are here considered as those who tolerate a full normal diet at the end of an arbitrarily established period of 3 years. Patients not cured were those who were unable to take a full diet at the end of 3 years. Of the 8 failures, 4 patients refused to follow the diet strictly and persistently; one was finally cured after 4 and 1/2 years; one was cured after 6 years; one suffered a relapse after asthma; and one, who had regularly eaten potatoes from the early months of treatment, now has loose stools whenever excessive amounts of candy or cake are eaten. (Postgrad. Med., April '50, S. V. Haas and M. P. Haas)

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Chemicals Introduced in the Production of Foods: This report summarizes some of the health problems involved in the use and control of chemicals introduced in foods. Foreign substances have been artificially introduced in foods for many years. The chemicals introduced in foods may become incorporated as a result of contamination from the use of pesticides or from contact with other substances, or they may have been added purposely in processing. The use of chemicals, so that they become contaminants, or serve as functional ingredients of processed foods, has increased greatly in recent years.

Although legislation has been enacted in most countries to effect control over practices which may be dangerous or deleterious to health, a consideration of some of the practices in the production and processing of foods shows that there are possible health or nutritional problems involved, which are not adequately controlled by methods currently available.

The use of inorganic poisons to control insect pests began with the development of Paris green for the control of the Colorado potato beetle, followed by the use of such materials as lead arsenate, particularly for the control of the codling moth in the growing of apples and pears. The use of lead arsenate as an insecticide in the growing of a number of crops was so increased, that the possible toxicity to agricultural workers and to consumers attracted increasing attention from health workers. The Federal Food, Drug, and Cosmetic Act of 1938 authorized the administrator of that act to establish limits for the quantities of harmful residues permitted in a food, provided it could also be shown that the use of the poisons was necessary in the production of the food. Tolerances have been established for residues containing lead and arsenic in certain foods. A tolerance for residues containing fluorine was invalidated by the courts, on a technicality.

The problem of the effective control of weeds, rodents, insects, and molds in the growing of foods is so grave that the search for more efficient pesticides continues. The number of important chemicals now being used in pesticides is probably about 25 or 30; but the possible number that may have usefulness is practically unlimited. The Federal Insecticide, Fungicide, and Rodenticide Act of 1947, administered by the U. S. Department of Agriculture, does not provide control over the sale and use of pesticides. For a number of years interest has been largely in the development of organic poisons, for it was formerly believed that among the organic compounds would be found effective pesticides which would also be relatively innocuous to higher forms of life.

Recent reports in the scientific literature show clearly that most if not all of the useful insecticides also have toxic properties for man. There have been reported 4 deaths from acute poisoning by parathion among workers engaged in handling the compound, and 3 additional cases of sudden death from the use of parathion by workers in the fields. The continued ingestion of DDT has been found to lead to the accumulation of DDT in the fatty tissues of experimental animals, and to the production of damage of the liver and nervous system. Cases of accidental acute poisoning from preparations containing DDT have been reported. Studies have shown that the application of DDT to alfalfa for the control of insects gives a higher yield and a higher content of carotene, but at the same time the alfalfa was also found to contain DDT. When fed to animals, this contaminated alfalfa led to the presence of DDT in the eggs and flesh of chickens, in the milk and beef of cows, and in the meat

of lambs. Benzene hexachloride, first thought to be relatively harmless to higher animals, now is known to have toxic properties similar to those of DDT. When applied to growing crops, it is subsequently detected in the tissues of vegetables such as potatoes, cucumbers, and beans. A recent study of chlor-dane showed that parenchymatous degeneration of the liver and kidney occurred in chronic poisoning.

More effective controls over residues of insecticides in some food articles will result no doubt from the recent public hearings of the Food and Drug Administration for the purpose of establishing permissible tolerances of poisons on or in fresh fruits and vegetables when it is shown that use of the poison is essential for the production of the food. Each pesticide will probably need individual consideration and final evaluation of possible effects on the health of consumers. Reliable practical methods of analysis for many of the pesticides still remain to be developed. Also needed is a simple but adequate set of instructions on the use of each pesticide concerning the dangers involved and the precautions to be exercised.

Chemicals are used in the production of foods for other purposes than pest control. Although lead arsenate has been found to accelerate the metabolism of some citrus fruits, oranges from treated trees are found to be markedly deficient in ascorbic acid. Doubt has already arisen concerning the possible effects of the administration of metabolic alteratives on the meat, milk, and eggs of farm animals. The administration of iodocasein to dairy cows causes an increased milk production. Thyroid depressant drugs, such as thiourea and thiouracil, have been shown to decrease the amount of feed needed to produce unit weight increases in hogs, and to improve the apparent market quality of broilers. The feeding of 3,4-dianisylhexene-3 to chickens is said to improve their market quality and the rate of fattening. Implantation of pellets of stilbestrol beneath the skin of chickens and turkeys has been reported to increase fat deposition and improve the quality of the meat.

The effects of metabolic alterations on the composition of animal foods should be defined before the use of such drugs becomes established. Although federal and state laws prohibit the incorporation of harmful or deleterious substances in foods, the burden of proof of toxicity at the present time often does not rest on the persons who want to use the material, but on the regulatory officials.

Substances purposely incorporated in foods during processing may be added for nutritional reasons or for functional purposes. Examples are iodized salt, and the enrichment of flour and bread with thiamine, riboflavin, niacin, and an assimilable iron preparation; traces of a thiosulfate may be added to iodized salt to help stabilize the iodide; tricalcium phosphate may be added to the same salt to confer on it "free-running" qualities. Occasionally,

the use of a functional ingredient results in the direct improvement of nutritional value of a food; the calcium content of the diet is improved through the use of small amounts of calcium salts in phosphated and self-rising flours and in other foods. Similarly, the vitamin C content of frozen peaches is improved when ascorbic acid is added to prevent browning of the cut fruit.

For the person who is obliged to adhere to a low-sodium diet, some salt substitutes have been proposed which impart a degree of salty flavor to foods, but which contain no sodium. It is now recognized that lithium salts are unsuitable as salt substitutes. Had lithium chloride been considered a drug it could not have been used in the dietary management of hypertension without going through the procedure of testing, evaluation, and licensing which is required of a new drug under the federal law before it can enter the channels of interstate commerce. Lithium chloride was considered to be a food and it was distributed without sufficient testing. Regulatory action could only be taken after convincing evidence of harmful effects was obtained.

Little can be done to prevent the addition of many new ingredients to an unstandardized food product unless evidence of their harmful nature is available, and of sufficient weight to hold up in court. The public is guarded chiefly by the awareness of food manufacturers; this protection is regarded as valuable, generally effective, and not to be underestimated. When experts disagree about the adequacy of evidence bearing on toxicity, difficulties may arise. Full information is sought by investigators in the Food and Drug Administration, but the problems are many, the staff and appropriations are limited, and time is required to obtain experimental results. Until data become available, considerable damage may be done. For example, monochloroacetic acid was described in a French patent in 1933 as a preservative of foods and beverages. A report later appeared in this country in which the authors concluded that monochloroacetic acid, in commercially useful quantities, was harmless even in infants. Later studies, chiefly of the Food and Drug Administration, showed that this substance was irritating to the gastro-intestinal tract. Subsequently, reports appeared of nausea and vomiting following consumption of beverages which had been stabilized with monochloroacetic acid. After these facts became known, hundreds of thousands of dollars worth of products containing this substance were caused to be destroyed. For the manufacturers who may suffer such losses, it may be evident that adequate testing of new ingredients, and approval by an unbiased group of scientists, is desirable before commercial use is made of any new ingredient in a processed food.

Thiourea, which will prevent the browning of cut sections of certain fruits, was originally reported to be harmless, but further studies revealed that thiourea is a goitrogenic agent. The Food and Drug Administration in 1946 effected a number of seizures of frozen peaches which, on analysis, were found to contain about 45 p.p.m. of thiourea as an antibrowning agent. Fortunately, experimental evidence of the toxicity of thiourea was obtained sufficiently

early to prevent extensive commercial use of the substance in human foods. Reports in the scientific and technical literature have called attention to the functional value of thiourea as an antioxidant for solutions of vitamin C, as an ingredient for the retardation of rancidity in certain dried milk products used in infant feeding, for the control of mold in moist wheat, and as an ingredient of a dip for the prevention of stem-end rot and decay by the blue and the green molds in oranges.

Mineral oil is prohibited as an ingredient of foods, even when declared on the label, because convincing evidence of its deleterious effects, when consumed with foods, is available. Mineral oil, when ingested with foods, preferentially dissolves carotene, and vitamins K and D, and prevents their absorption.

Since 7 August 1949, the use of nitrogen trichloride as a maturing and bleaching agent for flour has been prohibited. Even before that date, its use in domestic flour had become very small, as the result of voluntary action by the milling industry, following verified reports of the adverse effects on the nervous system of several species of animals to which flour heavily treated with nitrogen trichloride had been fed.

The Food and Drug Administration recently concluded public hearings for the purpose of receiving factual information about new optional ingredients offered for sale to bakers since 1943. Included among the optional ingredients, used not only by some bread bakers, but also used to some extent in the production of commercial cake, ice cream, mayonnaise, salad dressing, and other products, were a number of emulsifying or surface-active agents about the complete harmlessness of which several qualified experts expressed some doubt. Other witnesses voiced their approval of some of these chemicals whose chief function in bread production appears to be that of producing an unusual degree of softness of the loaf.

Apart from the question of toxicity, it has been shown that the use of some of the surface-active agents in processed foods permits reduction in the amounts of fat, milk products, and eggs which customarily have been used in these foods. So great a part of the food supply may now be in the form of packaged products that it is conceivable that marked alterations in the nature and nutritive quality of the diet might occur without the knowledge of consumers, and without the awareness of many professional persons interested in the maintenance of public health. Such possibilities should emphasize the need for careful evaluation of the probable effects of the use of any new chemical recommended for food use, prior to its use.

Although inspection of mortality records seem to show that accidental deaths from toxic chemicals of all kinds are an insignificant part of the total causes of death, the toxicologic properties of many of the chemicals used in

the production and processing of foods are unknown, particularly the chronic effects of their consumption in foods over a long period of time. Mistakes in judgment about the health qualities of chemicals introduced in foods may adversely affect the health of great numbers of persons. It does not seem that the consideration of preventive measures in this field should demand impressive statistics from the death records in order to justify the effective control of chemicals introduced in food processing and production.

The authors conclude that further study should be devoted to methods of detection of traces of foreign substances introduced in foods, and that every effort should be exerted to keep toxic substances at a minimum in those foods which cannot be produced without their use, and that their presence in other foods should be avoided and prohibited. Further research on all aspects, especially the medical problems, of chemicals introduced in foods should be encouraged.

More rigid control of new substances suggested for use in the production or processing of foods should be established. It would be advantageous to the food industries as well as to the general public if the present Federal Food, Drug, and Cosmetic Act were amended so that the same kind of authority presently granted over new drugs were made applicable to new chemicals, or new ingredients, intended for use in foods. When this has been done, a significant step in health protection will have been taken. (Am. J. Pub. Health, Year Book, Part II, May '50, from the Report of the APHA Committee on Chemicals Introduced in Foods, F. C. Bing, Chairman, et al.)

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Disposal of Radioactive Wastes in a General Hospital: The increasing use of radioactive isotopes in medicine has created several new problems of safety to the patient, the attending personnel, and the public. Some of these problems have to do with permissible doses from various sources, other with undesirable immediate and late effects, and still others with the handling of the numerous, usually very complex radioisotopes. It is important that certain minimal protective measures be taken to meet any situation that might arise. During the past few years considerable attention has been focused on the problem of disposal of wastes from general hospitals or research institutions using radioactive isotopes for diagnostic, therapeutic, or experimental purposes.

Very recently, Kenneth G. Scott made a study of the disposal of radioactive wastes at the Medical Center of the University of California in San Francisco, based upon data obtained in conjunction with the present therapeutic use of the most common radioisotopes. Scott found that when an institution houses 50 patients receiving ordinary radioisotope therapy, the activity of the

daily waste output may be as high as 1 curie per day. He estimated that if 20 of these 50 patients are treated with a probable dose of 100 millicuries of I^{131} , another 20 with a probable dose of 20 millicuries of P^{32} , and the remaining 10 with a probable dose of 100 millicuries of other fission products and radioactive rare earths, the average daily excretion per patient would amount to 50 millicuries for the radiiodine, 1 millicurie for the radiophosphorus, and 1 millicurie for the other fission products and radioactive rare earths. Thus, the total daily excretion for all patients would be 1,000 millicuries for the radiiodine, 20 millicuries for the radiophosphorus, and 10 millicuries for the other fission products and radioactive rare earths. If such highly concentrated radioactive material is admitted into the hospital sewerage, a dangerous situation may arise, particularly if the draining pipes contain many traps and pumps permitting the accumulation and storage of the radioactive isotopes for prolonged periods.

Scott also made some very enlightening estimates to illustrate the hazard that may accrue both inside and outside the hospital from the disposal of large amounts of radioactive wastes via the sewerage. To obtain more accurate figures, he assumed that no accumulation because of faulty or inaptly constructed plumbing fixtures takes place. If the excretion of 1 curie of activity referred to above were diluted in the total one-day sewage output of the University of California Medical Center, it would yield a gamma radiation corresponding to 2 mr. per day in liter quantities. A 5-cc. sample of such a solution would produce over 50 counts per second in a Geiger-Müller counter. The beta activity in the vicinity of the accessible ranges would have considerably higher values, a 5-cc. sample producing over 1,000 counts per second in a beta-sensitive counter.

The sewerage of most hospitals or institutions is connected directly with the public sewerage which is usually provided at its terminal with a filtering or processing plant. Scott calculated that the considerably greater dilution of the raw sewage at the San Francisco plant still would show an activity corresponding to about 100 counts per minute in a 5-cc. sample. The gamma-ray activity of such a solution would amount to less than 0.1 mr. per day. The filter cake, which is collected from the raw sewage, is used for economic purposes, chiefly as fertilizer. In this manner, radioactive wastes may be disseminated, contaminating fields, public parks, water pools, etc. Scott estimated that contact with a fertilizer made from a filter cake separated from radioactive raw sewage could lead to a skin exposure as high as 2 rep per day. This dose would fog a photographic film in a few hours.

It is probable that with further extension of the medical use of radioisotopes, in some localities more than 50 patients will be treated simultaneously in various hospitals. If all these hospitals use a disposal of the radioactive wastes via the public sewerage, an aggregate activity may result which is considerably larger than that estimated by Scott.

In 1949, the National Committee on Radiation Protection appointed a Subcommittee on Waste Disposal and Decontamination to carry out studies and make recommendations.

In the meantime, the Isotopes Division of the Atomic Energy Commission, has issued a circular giving interim recommendations for the disposal of radioactive wastes. These recommendations refer only to I^{131} and P^{32} which are the most commonly used isotopes in medical diagnosis and therapy and to C^{14} which is used mostly in research for labeling organic compounds. For radioiodine, the interim recommendations specify that the radioactive material or the radioactive wastes may be discharged into the public sewerage only under certain provisions. The first of these provisions postulates that the volume of water flowing from the hospital outlet into the public sewer must be sufficient to dilute the I^{131} to 0.5 microcurie per liter or, when 1 gram of potassium iodide is added to each millicurie of I^{131} at the time of disposal, to dilute it to 10 microcuries per liter. The second provision is that the maximum activity disposed of from any one institution shall not exceed 200 millicuries per week. For the radiophosphorus a dilution of 0.1 microcurie per liter is specified and in order to prevent marked reconcentration by any organism in the sewage it is recommended that each millicurie of P^{32} be diluted with 10 grams of phosphorus as phosphate at the time of the discharge. There are other recommendations applying in various situations or accidents in conjunction with the general handling of radioactive isotopes.

At the present time very few hospitals or institutions make use of such large doses of radioactive material as those indicated by Scott in his estimates. Likewise, the diagnostic tracer studies and experimental investigations are being conducted on a comparatively limited scale. The interim recommendations, which are subject to enlargement as indicated, therefore, are sufficient to meet the immediate requirements. The interim recommendations will be superseded by the recommendations, when made, of the Subcommittee on Waste Disposal and Decontamination. It is essential that general hospitals or institutions making use of radioisotopes strictly follow these recommendations. (Am. J. Roentgenol., April '50, Editorial)

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Bedside Determination of Chloride: A Method for Plasma, Urine, and Other Fluids and Its Application to Fluid Balance Problems; The author describes a bedside method for the estimation of chloride in plasma, urine, and other body fluids, and suggests some possible applications of this method to fluid balance problems.

The mercurimetric determination of chloride is an ideal basis for a simplified procedure because the endpoint is sharp, the titration is direct,

and substances such as protein and uric acid do not interfere. In this method, the pH of the sample must be controlled. It has been determined experimentally with a glass electrode that the most desirable pH is 1.8 or below, especially if the sample contains protein. The pH should never be above 2. The amount of acid used in this method has been determined to insure that the pH of the various samples always will be below 2. An acidified sample is titrated with mercuric nitrate in the presence of diphenylcarbazone. The mercuric nitrate reacts with the chloride in the sample to form soluble but unionized mercuric chloride. When all the chloride is used up, the excess mercuric ion gives a strong purple color with diphenylcarbazone.

Standard Sodium Chloride Solution, 100 mEq. per Liter: dissolve 5.85 Gm. of pure, dry sodium chloride (NaCl) in distilled water; make volume to one liter. Mercuric Nitrate: weigh out 17.13 Gm. of fresh mercuric nitrate ($\text{Hg}(\text{NO}_3)_2 \cdot \text{H}_2\text{O}$) in a small beaker. Add from 50 to 100 ml of distilled water and 1 ml of concentrated nitric acid. Stir until the salt is completely dissolved. Then make volume to 1 liter with distilled water. If it is desired that the results of the chloride determination be expressed in units other than milliequivalents per liter, the quantities of sodium chloride, mercury, and acid given in the table on the left

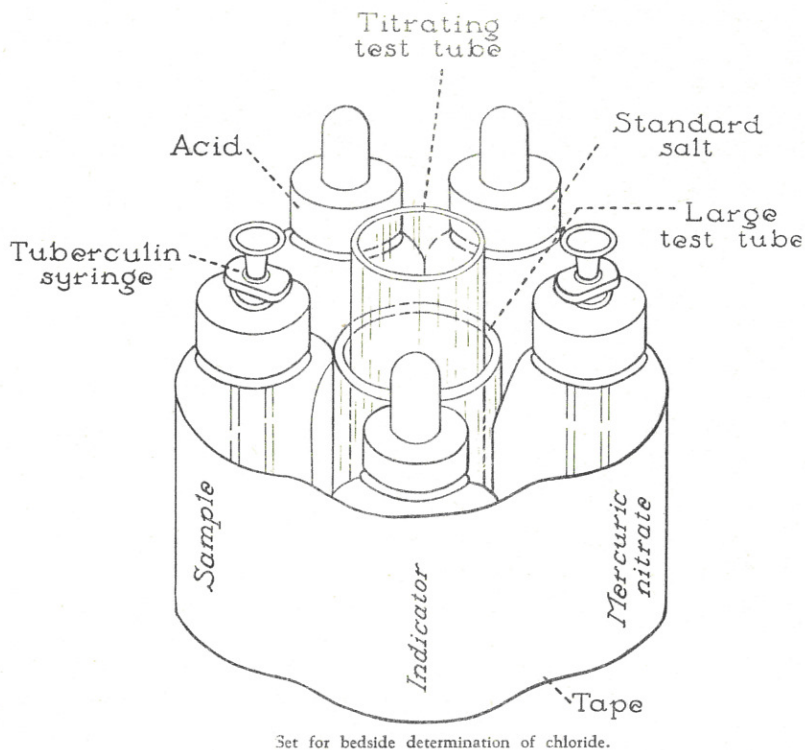
Units desired	Standard sodium chloride, gm. NaCl per liter	Mercuric nitrate, gm. $\text{Hg}(\text{NO}_3)_2 \cdot \text{H}_2\text{O}$ per liter	Concentrated nitric acid in mercuric nitrate, ml.
Milligrams sodium chloride (NaCl) per 100 ml.	10.00	29.30	2
Milligrams chloride (Cl^-) per 100 ml.	8.25	24.20	2

should be used. Indicator: dissolve about 400 mg. of diphenylcarbazone (Eastman Kodak No. 4459) in about 100 ml of 95-percent ethyl alcohol. Store in a light-tight bottle in the refrigerator. Make up fresh indicator at least every 6 months. Tenth Normal Nitric Acid (Approximate): dilute

7.0 ml of fresh concentrated nitric acid to one liter with distilled water. Supplies Other than Reagents. 1. Four 2-ounce (approx. 60 cc.) standard medicine dropper bottles with one-piece molded rubber caps (made by Armstrong Cork Co., and available at most drugstores). 2. Two 1-ml tuberculin syringes. One of these is to be used to measure the mercury solution, and must be carefully selected. Its plunger should stop exactly at the zero mark when pushed all the way down. Otherwise inconvenient corrections will have to be made during the titration. 3. One 1-ounce (approx. 30 cc.) standard medicine dropper bottle. A 2-piece plastic top is preferable to prevent sticking. 4. A test tube about 1 inch (2.5 cm.) in outside diameter and about 4 inches (10 cm.) long. 5. Another test tube about 3 inches (7.5 cm.) long, of diameter great enough to allow the 4-inch (10 cm.) test tube to slip inside.

Assembly of the Set. Make the 1-ounce bottle for indicator light-tight with tape and black paper. Remove the droppers and cut the bulbs away from the molded rubber caps of two of the 2-ounce bottles and slip the tuberculin syringes through the holes. The dropper is left in the cap of the bottle that is to contain acid, but is removed from the cap of the bottle that is to contain standard salt solution. Drop the test tube of smaller diameter into the test tube

of larger diameter. Group the bottles around the test tubes positioning them as shown below and tape all together. Label the bottles according to their con-



tents. Be sure that the specially selected syringe for mercury is in the mercury bottle. (Complete sets, including reagents, can be obtained from Rochester Products Co., Rochester, Minn.)

A little water should be kept in the bottle marked "sample" to prevent drying and sticking of the sample syringe. The indicator should be renewed every month from the supply which is kept in the refrigerator. The test tube of large diameter is provided only as a stand for the one of smaller diameter. In this latter tube, the titration is performed.

Handling of Blood Samples. Undue exposure of whole blood to air should be avoided by keeping the sample in a stoppered container until the plasma or serum is separated from the cells. A small portable centrifuge would be required to obtain plasma rapidly at the bedside. However, if the rate of sedimentation of erythrocytes of a patient's blood is high, plasma can be obtained in a few minutes simply by allowing the blood to stand. If time is not a factor, serum can be used after the clot has retracted.

Measuring of Solutions. In measuring solutions with tuberculin syringes, certain points of technic enhance the accuracy of the estimation: 1. When filling

a syringe, excess solution adhering to the outside of the syringe is removed by touching the tip to the container holding that solution. 2. After delivering solutions into the titrating test tube, touch off the last drop on the side of the tube. 3. Before a sample is measured for analysis, the syringe should be rinsed once with a small amount of that sample, although when there is insufficient sample, the error will probably not exceed ± 1 percent. 4. After a titration is complete, the outside of the mercuric nitrate syringe should be wiped free of sample to avoid diluting the reagent when the syringe is returned to its bottle.

Standardization of the Chloride Determination. Fill the sample syringe to the 1 ml mark with standard NaCl solution. Deliver this sample into the titrating test tube by pushing the plunger all the way down. Complete the analysis for chloride as directed below under Procedure. Note the volume of mercury reagent required to react exactly with the standard sodium chloride solution. Then readjust the volume of the sodium chloride sample so that 1.00 ml of mercury reagent will react exactly with it. For example, suppose 1.02 ml of mercury reagent is required to react exactly with the standard sodium chloride solution delivered from the 1 ml mark. On the next try, the standard sodium chloride sample should be delivered from about the 0.98 ml mark. Repeat this procedure until a plunger setting for the sample syringe is found such that the volume of standard sodium chloride solution delivered from this setting will react exactly with 1.00 ml of mercury reagent. A new setting should be determined for each new batch of mercury reagent or if new syringes are used. This setting is always used for measuring unknown samples. Standardization of the chloride method in this fashion eliminates errors inherent in weighing the mercuric nitrate and minimizes errors in the calibration and handling of the tuberculin syringes. Exactly the same procedure must be followed in titrating an unknown sample as was followed in titrating the standard sodium chloride solution.

The Endpoint. When the purple endpoint with some specimens is not as striking as with the standard NaCl solution, the first definite purple tint should be taken as the endpoint. This purple tint should not be confused with a salmon pink color that often precedes it. In hemolyzed or jaundiced plasma the endpoint color is reddish brown instead of purple. The endpoint color is often obscured by the pigment of the sample (such as bile or liquid stool), and has to be taken as a darkening of the solution rather than a definite color change.

Procedure. 1. Fill the sample syringe to the correct setting with the specimen to be analyzed. Deliver the specimen into the titrating test tube. 2. Add 5 drops of indicator. 3. Add 3 dropperfuls (between 2 and 2.5 ml) of tenth normal nitric acid. 4. Fill the mercuric nitrate syringe to the 1 ml mark. Place the syringe in the titrating test tube so that the molded rubber cap rests on the rim of the test tube. Hold test tube and cap with one hand, steadying the plunger with the index finger. Add the mercuric nitrate slowly

by pushing down the plunger with the other hand. Better control is obtained if the plunger is rotated as it is advanced. Agitate the sample by shaking the test tube and syringe as a unit. Add mercuric nitrate until the purple color appears. Add only a fraction of a drop at a time, as the endpoint is approached, by washing it off the syringe tip with the sample. 5. Each 0.01 ml of mercuric nitrate solution used in the titration is equivalent to 1 mEq. of chloride per liter, 10 mg. of chloride as NaCl per 100 ml or 5 mg. chloride as Cl^- per 100 ml, depending on how the mercuric nitrate solution was made up (see section on reagents and table). The result, therefore, can be read off the scale on the tuberculin syringe. The actual numbers on the scale, however, must be reversed in the mind of the user, because the zero point is at the distal end in the titration. For example, when the result of the titration reads 0.20 ml on the scale, actually 0.80 ml of mercury reagent has been used. 6. Without changing the amount of indicator or tenth normal nitric acid, the volume of the sample can be cut to a half or a fourth. The results are then doubled or quadrupled, and the accuracy is somewhat less than when the larger volume is used.

Accuracy of Method. The method is accurate to ± 1 percent on duplicate determinations of plasma chloride, and can be improved with practice. With the exception of one sample (2.9 percent high), results on 20 consecutive samples of plasma agreed within a range of ± 1.9 percent with results obtained by the silver nitrate method of Wilson and Ball. The accuracy of determination for urine chloride was comparable but decreased as the concentration of chloride decreased. Thus, on analysis of 14 specimens of urine in which the concentration of chloride was more than 50 mEq. per liter, the results were all within plus 2.2 percent to minus 1.0 percent of results obtained with the Wilson-Ball method. On 6 specimens of urine in which the concentration of chloride was less than 50 mEq. per liter, the errors ranged from plus 7.1 percent to minus 0.5 percent. Satisfactory results also were obtained on a variety of other fluids as shown in the table on the left.

Comparison of Results Obtained by the Bedside Method for the Estimation of Chloride in Various Fluids With Those Obtained by the Chloride Method of Wilson and Ball.⁵ Each Specimen is From a Different Patient. Results Are Expressed in Milliequivalents per Liter

Fluid	Bedside method	Wilson-Ball method	Difference, per cent
Bile from common duct	100.0	98.4	+1.6
Duodenal aspiration	66.0	67.6	-2.4
Ileac content	116.0	116.8	-0.7
Ground up and liquefied food	30.0	31.0	-3.2
Gastric juice	127.0	126.8	+0.2
Gastric juice	118.5	120.2	-1.4
Liquid stool	55.0	54.2	+1.5
Duodenal aspiration	78.0	75.5	+3.3
Bile from common duct	98.0	100.0	-2.0
Ileac content	90.0	88.4	+1.8

Application of the Determination to Problems of Fluid Balance.

Because the determination of chloride by the method described is so rapid and inexpensive, frequent analyses of plasma chloride can be made and the results can be used to help in the management of problems of fluid balance. However, the value for plasma chloride often is difficult to interpret unless the value for plasma bicarbonate also is known. A bedside-bicarbonate method is being developed that can be performed on the same sample as that used for estimation of chloride.

Because this quantitative test for chloride can be performed quickly at the bedside on any liquid specimen, it makes practical a new approach to the clinical management in problems of fluid balance, i.e., the determination of the intake and output of chloride as well as water from which a daily water-chloride balance sheet can be constructed.

How to Make a Water-Chloride Balance Sheet. All urine, liquid stool, gastric fluid and so forth are saved in marked containers and are measured and analyzed daily to determine total volume, chloride and pH. Total intake of water and chloride also are noted. From these data a daily balance sheet as shown below is constructed.

A WATER-CHLORIDE BALANCE SHEET

From: 10 a.m. 11/21/49 To: 8 a.m. 11/22/49 Equals: 22 hours

INTAKE*			OUTPUT				
Volume (ml.)	Type of Fluid	Total Chloride (mEq.)	Volume (ml.)	Type of Fluid	Conc. of Chloride (mEq./L.)	Total Chloride (mEq.)	pH**
3,000	5 per cent dextrose in H ₂ O, parenteral	0	1,200	Urine	34	41	5.5
3,000	5 per cent dextrose in 0.9 per cent NaCl, parenteral	462	1,000	Insensible and sensible loss†	—	0‡	—
			2,100	Gastric aspirate	5	120	1.5
200	Oral fluid	0	1,700	Ileac content	114	194	7.0
6,200	Totals	462	6,000	Totals	—	355	—

*The water of oxidation is neglected; it probably averages around 250 ml. per 24 hours.²⁰

**pH is determined by the use of indicator paper. The paper used for this purpose is "Accutint Wide Range B" made by Anachemia Ltd., Montreal, Canada. However, a rough estimate of the pH can be obtained by noting the color of the sample when diphenylcarbazone is added. The sample will turn yellow when the pH is below 5; orange when the pH is between 6 and 7; and cherry red when the pH is above 8.

†The following guide has been found helpful in obtaining a rough estimate of 24-hour sensible and insensible loss: No fever and no perspiration—1,000 ml.; high fever or moderate perspiration—1,500 ml.; perspiration enough to require change of bed clothes—2,000 ml.; higher volumes for excessive perspiration.

‡Negligible chloride is lost with insensible perspiration.¹¹ One can estimate that 70 mEq. of chloride is lost with every 1,000 ml. of perspiration in excess of 1,000 ml.¹² Thus if the loss was estimated at 3,000 ml. the chloride loss would be estimated at 140 mEq.

A nurse can be trained easily to make the chloride determinations and to construct a balance sheet. One person so trained can make daily balance sheets in all the fluid balance problems in a hospital. The time required to measure and analyze the material, assemble the data, and construct a balance sheet, averages about 30 minutes.

Application of Balance Sheet. A patient may retain several hundred milliequivalents of sodium and chloride without developing edema. A balance sheet gives early warning of overdosage with sodium chloride long before edema develops, by indicating a markedly positive chloride balance. A negative chloride balance affords similar protection against a sodium chloride deficit long before it becomes detectable clinically. If desired, the actual changes in the volume of the extracellular (chloride) space can be calculated

from the chloride balance. In addition, daily determinations of urine chlorides, which in themselves can give useful information regarding electrolyte balance, are provided. Disturbances of acid-base balance often can be anticipated by noting the pH of the various specimens. For example, if a large volume of gastric juice is lost and its pH is 1.5, alkalosis might be suspected.

The simplest and perhaps the most useful application of the water-chloride balance sheet is its use in planning administration of fluids from day to day. By noting the figures representing the output of water and chloride for the day preceding, one can estimate the output for the next day and plan treatment accordingly. Experience with the management in fluid balance problems by the aid of a water-chloride balance sheet suggests that better care can be provided at lower cost. Fewer errors are made in treatment and the number of blood chemical studies reduced. The management of patients with fluid balance problems becomes more exact and instructive. (Proc. Staff Meet., Mayo Clin., 26 April '50, B. H. Scribner)

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Effect of Free and Combined Available Residual Chlorine upon Bacteria in Swimming Pools: Sanitary control of swimming pools requires the maintenance of some fixed amount of residual chlorine, which will kill bacteria, as they are washed into the water from the bathers' bodies, rapidly enough to prevent conditions of gross pollution. In an attempt to standardize the magnitude of this fixed amount of residual chlorine, inconsistent bactericidal results were frequently obtained when a standard quantity of residual chlorine was used. These findings were probably caused by the alternate presence of the 2 components of total residual chlorine, i.e., free available residual chlorine and combined available residual chlorine. In 1942, the joint committee of the Conference of State Sanitary Engineers and the American Public Health Association issued a report recommending a residual chlorine concentration of from 0.4 p.p.m. to 0.6 p.p.m. when the pool was in use, and of from 0.7 p.p.m. to 1.0 p.p.m. for residual chloramine. This latter figure was modified in 1948 to suggest the desirability of operating with higher chloramine residuals up to 2.0 p.p.m. The term, chloramine residual, has been changed to combined available chlorine residual, by the American Water Works Association committee which prepared the chapter on "Chlorination and Other Disinfection Practices" for the forthcoming Manual of Water Quality and Treatment.

The development of the orthotolidin-arsenite colorimetric test and the amperometric titration method for determining residual chlorine gave the sanitary engineer quick, efficient, and accurate methods for measuring in the field the quantities of free and combined available residual chlorine present in water. With these resources available, a study was undertaken to measure the relative effectiveness of free and combined available residual chlorine as bactericides

in swimming pool water. To obtain this measurement it was decided to use tests for the number of bacteria which grow on agar at from 35 to 37° C., coliform bacteria, and streptococci. The first 2 tests are in common usage in sanitary bacteriology, but the latter test is not widely used in this country. There has been some disagreement concerning the usefulness of the streptococcal density in evaluating the sanitary quality of swimming pool water.

For this study, four pools in Connecticut were selected. Each pool was operated in accordance with the requirements of the Joint Committee Report and was of the continuous recirculation type. All pools used fresh water and 3 were located indoors. Although combined residual chlorination through the use of ammonia gas was not the practice by intention, one pool applied aluminum ammonium sulfate and actually obtained this method of disinfection and a second pool changed to it accidentally during the test period by changing coagulants.

Each of the 4 pools was tested on 3 occasions, making a series of 12 tests within this study. Before each test, the residual chlorine content was measured to insure reasonable adherence to the minimum and maximum standard for disinfection as set forth in the Joint Committee Report. The amperometric titration was used in preference to the orthotolidin-arsenite test to measure free and combined available residual chlorine, because the high temperatures of swimming pool waters cause the color reaction of chlorine and orthotolidin to occur too quickly for accurate results.

From this study, the following general conclusions were reached:

Free available residual chlorine is a more effective bactericide than combined available residual chlorine for the treatment of swimming pool water. The values of from 0.4 to 0.6 p.p.m. of residual chlorine as recommended by the Joint Committee Report, if interpreted to mean free available residual chlorine, will produce bacteriological results that will comply with the Joint Committee Report's recommended standards for total number of bacteria and coliform bacteria in swimming pools when in use. The minimum value of 0.7 p.p.m. which has been suggested in the past for swimming pools using chloramine (combined available residual chlorine) treatment is insufficient to produce bactericidal results under conditions of this study which will meet the recommended standards of the Joint Committee Reports. Streptococcal bacteria in swimming pools are more resistant to chlorine than are coliform bacteria. (Am. J. Pub. Health, April '50, E. W. Mood)

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Interim Recommendations for Treatment in Fluoroacetate Poisoning:

The selection of the best antidote for fluoroacetate poisoning is complicated by the fact that there is little evidence of the effectiveness of any measure in man; experimental animals differ widely in their response to the poison; and some of the substances known to be effective, at least in experimental animals, are not generally available. On the basis of existing information the following interim recommendations are made:

1. Lavage the stomach to remove the contents. This measure may be of some value even several hours after the ingestion of the poison.
2. Administer orally 4 cc. per Kg. of an equal mixture of 50 percent ethyl alcohol (whiskey) and 5 percent acetic acid (vinegar), or either one alone if both are not available. This may be repeated in 3 or 4 hours, if necessary, or smaller doses may be given at more frequent intervals.
3. If convulsions occur, administer intravenously with great care small doses of a short acting barbiturate. There is evidence that fluoroacetate potentiates the depressant action of barbiturates.
4. The intravenous administration of plasma may be of value. Large quantities should be avoided because of the danger of adversely affecting cardiac function. The recommended dose is 5 cc. per Kg. of body weight.
5. If monoacetin (glyceryl monoacetate) is available, it may be substituted for the alcohol and vinegar regimen. It should be given intramuscularly, although it is irritating by this route. The pharmacologic effects of this agent are not known completely, although its acute toxicity is undoubtedly low. The dose recommended on the basis of animal experimentation is 0.25 cc. per Kg. every hour. In view of its effectiveness in animals, the availability of monoacetin is desirable in installations where fluoroacetate is employed.

The value of monoacetin as an antidote is probably a result of the generation from this compound of a utilizable acetate moiety. The administration of acetate per se, or as vinegar, provides a more direct, but an apparently less usable source of acetate. For this reason the co-administration of alcohol has been suggested to catalyze the oxidation of acetate. In addition, the alcohol is converted to acetate.

Because cardiac function is a prime consideration in fluoroacetate poisoning, frequent electrocardiograms provide an important diagnostic and prognostic aid. (Prepared 20 April '50, by the Physiology-Pharmacology Subcommittee of the National Research Council Chemical-Biological Coordination Center)

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Course of Instruction in Food Management for Officers of the Medical Service Corps, Regular Navy: The Bureau of Medicine and Surgery announces the establishment of a training program in Food Management at Cornell University, Ithaca, New York. Courses of one and two academic years in duration are open to officers of the Medical Service Corps in all ranks who have permanent status in the Corps. It is desirable that applicants requesting this instruction have had previous experience in commissary management and have completed the period of training offered at the U. S. Naval School of Hospital Administration, National Naval Medical Center, Bethesda, Maryland. It is anticipated that this program will remain in effect until the Medical Department has a sufficient number of well qualified officers in this field of administration.

In requesting this instruction it is necessary that the applicant submit in writing an agreement not to resign and to remain in the naval service for a period of 3 years, which includes the period of instruction, for the one-year course and 5 years, which includes the period of instruction, for the two-year course. Requests must be received in BuMed prior to 15 July 1950 in order to receive consideration and may be made by dispatch should the time element involved require such action. Dispatch requests must be confirmed by a following letter. (Professional Div., BuMed)

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Annual X-ray Study of the Chest: The Annual Sanitary Report of the Atlantic Fleet for 1949 indicates that annual x-ray studies of the chest had been made on 96 percent of the personnel aboard the ships of the fleet during December 1949. This is a creditable record, particularly in view of the fact that it was stated that two thirds of the personnel reporting aboard one of the vessels during the year were overdue for the annual x-ray; this in spite of the fact that a majority of the men came from shore stations where x-ray facilities were readily available.

Shore activities are urged (1) to insure that all personnel receive an annual chest x-ray examination, using the photofluorographic technic if possible, and (2) to examine without fail those men destined for duty with the fleet or on foreign soil who have not had an x-ray film study of the chest within the previous 6 months. (Preventive Medicine Div., BuMed)

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BUPERS CIRCULAR LETTER 50-60

25 April 1950

To: All Ships and Stations

Subj: Physical Standards for Enlistment

1. Effective immediately, physical standards for enlistment and re-enlistment in the Regular Navy and Naval Reserve of male applicants are modified as follows:

a. Dental requirements: Applicants must be well-nourished and have good musculature, be free from gross dental infections, and have a minimum requirement of an edentulous upper jaw and/or an edentulous lower jaw corrected or correctible by a full denture or dentures.

b. Vision: Visual acuity without glasses not less than 2/20 in each eye if vision is correctible to 20/20 in each eye and provided the defective vision is not due to active or progressive organic disease. Color-perception test will be administered for record purposes only. Defective color perception is not disqualifying.

c. Height: Minimum 60 inches, maximum 78 inches.

2. The foregoing modifications temporarily suspend specific corresponding standards of respective sections of part 2, Manual of the Medical Department. The possibility of subsequent disability claim will be considered in examining applicants with questionable defects. All minor and questionable defects will be recorded.

--BuPers. J. W. Roper

Approved: 27 April 1950
Francis P. Matthews,
Secretary of the Navy.

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BUMED CIRCULAR LETTER 50-53

15 May 1950

From: Chief, Bureau of Medicine and Surgery
To: All Ships and Stations Having Medical or Dental Officers Attached,
Except U. S. Naval Hospitals

Subj: Medical and Dental Periodicals for Ships and Stations: Subscriptions to

1. Ships and stations having a medical officer attached and now receiving only the Journal of the American Medical Association will also receive the periodicals

listed in items (a), (b), and (c) below. Item (d) will also be sent to stations caring for dependents of naval personnel. Ships and stations having a dental officer attached and not now receiving a dental journal other than the Journal of the American Dental Association will receive item (e):

- a. Surgery, Gynecology and Obstetrics
- b. Annals of Internal Medicine
- c. Postgraduate Medicine
- d. Archives of Pediatrics
- e. Oral Surgery, Oral Medicine and Oral Pathology

2. In case of nonreceipt of these periodicals addressees are requested to notify the Bureau of Medicine and Surgery.

3. It is desired that at shore stations, wherever feasible, copies of these periodicals be bound annually and retained. C. A. Swanson

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BUMED CIRCULAR LETTER 50-54

25 May 1950

From: Chief, Bureau of Medicine and Surgery
To: All Ships and Stations

Subj: Precious Metal Expenditures Reported on NAVMED Form L -
Report of Prosthetic Dental Treatment

Refs: (a) BuMed C/L No. 47-90 of 14 July 1947; AS & SL July-Dec 1947, 47-638, p. 233
(b) BuMed C/L No. 50-1 of 11 Jan 1950; N.D. Bulletin of 15 Jan 1950, 50-18.

1. Reference (a) is hereby canceled and superseded.

2. Reports of Prosthetic Dental Treatment containing precious metal value entries shall reflect average unit prices derived in accordance with reference (b). C. A. Swanson

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